Amniotic Fluid Embolism
An Obstetric Emergency

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Nadine C. Englert, RN, MSN

Although the first reported case of amniotic fluid embolism (AFE) was documented in 1926,1 a historic event that received much public and medical attention predates that case by more than 100 years. Public records indicate that in 1817 an obstetrician named Sir Richard Croft was widely criticized because of the unexpected death of one of his patients and her unborn son. The patient was Princess Charlotte of Wales, and the condemnation and grief Croft experienced because of this loss apparently led him to commit suicide. Investigations, continuing at least until the 1970s, strongly suggest that the princess died of AFE, possibly absolving Sir Richard of mismanagement of a difficult labor and highlighting a senseless third tragedy.2

Even today, AFE is the leading cause of death during labor and the first few postpartum hours,2 and it remains a deadly and unpreventable obstetric emergency.4 Despite technological advances in critical care life support, the maternal mortality rate for AFE remains around 61%; a large percentage of survivors have permanent hypoxia-induced neurological damage. The fetal mortality rate, although better than the maternal rate, is a dismal 21%, and 50% of the surviving neonates experience permanent neurological injury.5

Description and Etiology

Normally, amniotic fluid does not enter the maternal circulation because it is contained safely within the uterus, sealed off by the amniotic sac. AFE occurs when the barrier between amniotic fluid and maternal circulation is broken and, possibly under a pressure gradient, fluid abnormally enters the maternal venous system via the endocervical veins, the placental site (if placenta is separated), or a uterine trauma site.6 Why this entry into maternal circulation occurs in some women and not in others is not clearly understood. The devastating consequence of circulating fetal debris (carried by amniotic fluid) occurs only rarely, even though during normal labor and delivery, cesarean deliveries, and minor traumatic procedures fetal tissue may pass into maternal circulation and cause no symptoms.7 That AFE develops in only a minute proportion of these women suggests that either it is an effect of the amount of exposure to fetal debris or the type of fetal debris (containing meconium or not) or that some maternal factors may play a significant role.5,8 Clark et al5 contend that AFE more closely resembles an anaphylactic reaction to fetal debris than an embolic event, and they propose the term "anaphylac-
toid syndrome of pregnancy” instead of AFE. The exact mechanism of this anaphylactoid reaction to amniotic fluid is not clearly understood.

Predisposing factors once considered to be associated with AFE include placental abruption, uterine overdistention, fetal death, trauma, tumultuous or oxytocin-stimulated labor, multiparity, advanced maternal age, and rupture of membranes.4 However, in numerous documented cases of AFE, none of these conditions or demographic characteristics occurred or were applicable at the time of the event. Furthermore, a recent analysis of 46 verified cases of AFE did not substantiate most of these conditions as associated factors; 12% of the cases occurred in women with intact membranes, 70% during labor, 11% after vaginal delivery, and 19% during cesarean delivery with or without labor.5

**Incidence and Inclusion Criteria**

The reported incidence of AFE varies widely because of the obscurity of the problem and the wide range of signs and symptoms associated with it. According to Gilbert and Danielsen,6 AFE occurs in 1 of every 20 646 deliveries. Thanks to increasing awareness of the syndrome, this number is probably more accurate than many earlier estimates, which ranged from 1 in 8000 to 1 in 80 000. Precise reporting of incidence is extremely difficult because no definitive clinical or laboratory test is available that can be used to provide an exclusive diagnosis of AFE or completely rule it out. Clark and coworkers established a national registry for AFE in an effort to investigate and understand this syndrome more completely. Table 1 lists the inclusion criteria for diagnosis of AFE.

**Clinical Features and Pathophysiology**

One of the major factors that makes AFE so devastating is its total unpredictability. Although most cases occur after the onset of labor, some incidents have occurred outside of labor. As mentioned earlier, several clinical conditions seem to be associated with AFE, but essentially there are no definitive clues, warning signs, or associated conditions that indicate the risk of AFE may be increased. Most experts agree that AFE, as it is known now, cannot be predicted or prevented.11

According to Gei and Hankins,11 the initial signs and symptoms have a typical chronology, and the morbidity and mortality of the manifestations steadily decreases with time. Most often, respiratory distress and cyanosis occur suddenly within the first minute and are quickly followed by hypotension, pulmonary edema, shock, and neurological manifestations such as confusion, loss of consciousness, and seizures. More than 80% of patients experience cardiopulmonary arrest within the first few minutes.6 Approximately 50% of patients do not survive this onslaught of cardiopulmonary injury, but of those who do, 40% to 50% have coagulopathy and hemorrhage up to 4 hours later.12 Porter et al11 noted that in some patients, coagulopathy may be the first indication of AFE, whereas Clark et al reported that seizure activity may at times be the first manifestation.

The pathophysiology of AFE is speculative; various theories have been published. Gei and Hankins11 proposed a pathophysiological course (see Figure). Three distinct responses or a combination of clinical responses to circulating fetal debris are suggested.

The initial respiratory reaction possibly begins with transient pulmonary vasospasm.14 Although this possible transient vasospasm has not been documented (probably because the signs and symptoms appear so abruptly in a seemingly healthy person who is not being monitored via invasive methods), vasospasm may be caused by amniotic microemboli that trigger the release of arachidonic acid metabolites15 and lead to pulmonary hypertension, intrapulmonary shunting, bronchoconstriction, and severe hypoxia.11 Exactly which components of amniotic fluid actually cause this effect is unknown, but Clark14 suggests that abnormal components

**Table 1**

National registry’s criteria for diagnosis of amniotic fluid embolism 5,10

<table>
<thead>
<tr>
<th>Condition</th>
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<tbody>
<tr>
<td>Acute hypotension or cardiac arrest</td>
</tr>
<tr>
<td>Acute hypoxia, defined as dyspnea, cyanosis, or respiratory arrest</td>
</tr>
<tr>
<td>Coagulopathy, defined as laboratory evidence of intravascular consumption or fibrinolysis or severe clinical hemorrhage in the absence of other explanations (although patients who died before assessment of coagulopathy are eligible)</td>
</tr>
<tr>
<td>Onset during dilatation and evacuation, labor, cesarean delivery, or within 30 minutes postpartum</td>
</tr>
<tr>
<td>Absence of any other significant confounding condition or potential explanation of symptoms and signs just listed</td>
</tr>
</tbody>
</table>
Pathophysiology of amniotic fluid embolism.
Reprinted from Gei and Hankins, with permission.

Sequence of events:
1. For reasons not completely understood, amniotic fluid reaches the maternal intravascular compartment (systemic venous system).
2. The amniotic fluid enters pulmonary circulation via the pulmonary artery. This contaminated blood crosses to the left atrium through a patent foramen and probably through intrapulmonary shunts once the embolism is significant, as shown in cases of massive amniotic fluid embolism at autopsy.
3. The exposure of the pulmonary vasculature to both soluble (leukotrienes, surfactant, thromboxane A2, endothelin, etc.) and insoluble components (squames, vernix, hair, mucin, etc.) of the amniotic fluid and possibly other mediators released locally induces capillary leak, negative inotropism, and bronchospasm. This results in sudden onset of respiratory distress and cyanosis.
4. Within minutes, the negative inotropic effect becomes prevalent (probably due to myocardial ischemia). Pulmonary venous pressure (congestion) increases and a drop in cardiac output are manifested by pulmonary edema and hypotension to the point of shock.
5. The exposure of the intravascular compartment to amniotic fluid thromboplastin and to other mediators freed in the circulation by the presence of amniotic fluid induces a consumptive coagulopathy in a large proportion of the first-phase survivors. This disseminated intravascular coagulation often results in severe uterine bleeding.
6. The resultant systemic hypotension decreases the uterine perfusion. Abnormalities of the fetal heart tracing will rapidly follow and may result in fetal death.

Pathophysiology of amniotic fluid embolism.
Reprinted from Gei and Hankins, with permission.
between successive heartbeats as measured by the R-R interval of the QRS cardiac cycle and is reliably assessed only via internal fetal monitoring. Short-term variability gives the fetal heart rate tracing a zigzag appearance on the monitor strip.) A decrease in fetal oxygenation, in this situation due to maternal hypotension and hypoxia, can rapidly lead to the appearance of nonreassuring patterns in fetal heart rate (Table 2).

Correction of the cause of these changes, that is, maternal hypotension and hypoxia, is the only remedy. Therefore, resuscitation of the mother and stabilization of her condition are the priorities. When fetal heart rate is being assessed, the appearance of nonreassuring patterns should be viewed in the context of the overall clinical picture. Each of the patterns listed in Table 2 may have more than a single cause; some of the causes are relatively benign and easily correctable. For example, simply changing the mother’s position, providing her with fluids for volume expansion, and/or initiating oxygen supplementation may improve matters. However, the seriousness of AFE is quickly made evident by the rapid deterioration in fetal status and the apparent futility of measures normally implemented to improve it.

**Diagnosis**

Immediate recognition and diagnosis of AFE is essential to improve maternal and fetal outcomes. Until recently, the diagnosis of AFE was made only after an autopsy of the mother revealed squamous cells, lanugo hair, or other fetal and amniotic material in the pulmonary arterial vasculature. Although laboratory data may indicate that AFE is likely, as noted previously, no single laboratory or clinical finding can be used to diagnose or exclude it. Diagnosis, therefore, must be based on clinical features, and AFE should not be confused with other pregnancy-related complications or medical conditions (Table 3). Obstetric nurses and critical care nurses caring for obstetric patients should familiarize themselves with this condition, as Gei and Hanks imply that an increased awareness of this syndrome has resulted in earlier diagnosis, aggressive intervention, and probably a better chance of survival.

**Management**

Once the signs and symptoms are recognized and a presumptive diagnosis is made, supportive measures should be implemented promptly. In the event of cardiac arrest, the resuscitation team should follow standard Advanced Cardiac Life Support protocols for obstetric patients. Ideally, overall management of AFE should take place in an obstetric intensive care unit. Many hospitals lack obstetric intensive care units and so the patient must be cared for in a medical or surgical...
started at 5 cm H2O and increased by increments of 2 to 3 cm H2O until satisfactory levels of PaO2 are reached. The goal of oxygen therapy is to maintain arterial PaO2 higher than 60 mm Hg and arterial oxygen saturation at 90% or higher.

Circulation
Maintaining cardiac output and blood pressure involves several simultaneous interventions. Gei and Hankins recommend positioning the patient flat or in a slight Trendelenburg position to improve the venous blood return and perfusion of the central nervous system. Supportive measures include the initiation of fluid therapy, administration of pharmacological agents (Table 4), and electrocardiographic monitoring to detect and treat arrhythmias; most patients with AFE initially have electromechanical dissociation or bradycardia. Placement of a pulmonary artery catheter is highly recommended for monitoring cardiac output, central venous pressure, and pulmonary artery pressures (Table 5). In addition, pulmonary artery catheters provide direct access for blood samples to be sent for cytological analysis for amniotic fluid and fetal debris.

Oxygenation
The fetus is very vulnerable to maternal hypoxia, which is initially profound in AFE. Therefore, the first priority is resuscitation of the mother and administration of oxygen by any means available at concentrations of 100%.

A more aggressive approach includes securing an airway through endotracheal intubation, providing mechanical ventilation for the patient with a high inspired fraction of oxygen (≥60%), and the addition of positive end-expiratory pressure. Positive end-expiratory pressure is typically toward improving inotropy. Gillie and Hughes recommend several inotropic agents as drugs of choice for maintaining cardiac output and blood pressure (Table 4). A clinical guideline for supporting critically ill obstetric patients is to maintain systolic blood pressure at or higher than 90 mm Hg, with acceptable organ perfusion, as indicated by a urine output of 25 mL/h or more.

Control of Hemorrhage and Coagulopathy
The results of hematologic studies can be used to diagnose and monitor the course of bleeding and disseminated intravascular coagulation. According to Martin et al., control of uterine bleeding can often be accomplished with uterine massage and the administration of pharmacological agents (Table 4). If bleeding is profuse and pharmacological intervention is unsuccessful, a hysterectomy may be necessary.

Administration of blood transfusions and blood components is considered the first line of treatment for correcting coagulopathy associated with AFE. Blood products include packed red blood cells, fresh-frozen plasma, platelets, and cryoprecipitate to maintain organ perfusion and urinary output until bleeding due to disseminated intravascular coagulation resolves. Cryoprecipitate is particularly useful in AFE because it can be used to replenish clotting factors in lieu of fresh-frozen plasma in volume-restricted patients. In addition, cryoprecipitate contains both fibrinogen and fibronectin, which facilitate the removal of cellular and particulate matter (eg, amniotic fluid debris) from the blood via the reticuloendothelial system.
McCance and Huether explain that the reticuloendothelial system, now referred to as the mononuclear phagocyte system, consists of a line of cells that ingest and destroy (by phagocytosis) unwanted materials in the blood.

<table>
<thead>
<tr>
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<th>Pharmacological action</th>
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<td><strong>Inotropic agents to maintain cardiac output and blood pressure</strong></td>
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<td><strong>Dopamine</strong></td>
<td>Initial intravenous infusion: 2-5 µg/kg per minute Increase in increments of 5-10 µg/kg per minute as needed</td>
<td>Stimulates β₁-adrenergic receptors; causes release of epinephrine, which increases myocardial contraction, cardiac output, and stroke volume</td>
<td>Dopamine is a potent drug; be sure to dilute before administration; dopamine should never be given as a bolus dose</td>
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<td><strong>Dobutamine</strong></td>
<td>Intravenous infusion: 2-40 µg/kg per minute Rate of administration depends on patient’s response, including heart rate, blood pressure, cardiac output, and urine output</td>
<td>Stimulates β₁-adrenergic receptors; increases cardiac function, cardiac output, and stroke volume</td>
<td>Reconstitute solution according to manufacturer’s directions; reconstitution requires 2 stages</td>
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<td><strong>Norepinephrine (Levophed)</strong></td>
<td>Intravenous infusion: 2-4 µg/min Effect on blood pressure determines dosage</td>
<td>Stimulates α-adrenergic receptors; produces vasoconstriction and increased blood pressure; moderate increase in myocardial contraction caused by stimulation of β₁-adrenergic receptors</td>
<td>Continue infusion until blood pressure is maintained without therapy; avoid abrupt withdrawal</td>
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<td><strong>Oxytocic agents used to control uterine atony and bleeding</strong></td>
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<td><strong>Oxytocin (Pitocin)</strong></td>
<td>Dilute 10-40 U in 1000 mL isotonic sodium chloride or dextrose solution Titrated to control uterine atony, usually 0.02-0.1 U/min 0.2 mg intramuscularly or intravenously every 2-4 hours</td>
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<td><strong>Methylergonovine (Methergine)</strong></td>
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<td>Stimulates the rate, tone, and amplitude of uterine contractions and decreases uterine bleeding</td>
<td>Administer intravenously slowly over 1 minute; check vital signs for evidence of shock or hypertension after intravenous administration Because of hypertensive response, use cautiously with norepinephrine</td>
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<td><strong>Prostaglandin F₂ (PGF₂) (Hemabate)</strong></td>
<td>250 µg intramuscularly May be repeated every 15-90 min as needed for bleeding</td>
<td>Stimulates uterine contractions and decreases bleeding</td>
<td>A major contraindication to use is asthma, which may be exacerbated because of the drug’s bronchoconstrictive properties</td>
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<td><strong>Misoprostol (Cytotec)</strong></td>
<td>1000 µg rectally</td>
<td>Synthetic prostaglandin E₁; stimulates uterine contractions and decreases bleeding</td>
<td>Limited studies are available on the clinical efficacy of misoprostol in treating postpartum hemorrhage</td>
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<td><strong>Steroids to control inflammatory response</strong></td>
<td>500 mg intravenously every 6 hours</td>
<td>Pharmacological doses have marked anti-inflammatory effect because of their ability to inhibit prostaglandin synthesis</td>
<td>Administer direct intravenous solution at a rate of 100 mg/30 s; doses larger than 500 mg should be infused over 10 minutes</td>
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### Table 4  Pharmacological support in amniotic fluid embolism

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Another pharmacological intervention is the use of intravenous steroids. Amniotic fluid not only displaces blood and reduces oxygen and waste exchange but also introduces antigens, cells, and protein aggregates that trigger inflammation within the bloodstream. In consideration of the potential inflammatory response and similarities to anaphylaxis, the administration of corticosteroids (Table 4) may be helpful in AFE.

In summary, the 3 main goals of treatment are (1) oxygenation, (2) maintaining cardiac output and blood pressure, and (3) correcting coagulopathy. Intensive care nurses, including those without obstetric training, are expected to learn the critical assessment-management actions beginning with the primary and secondary surveys of critically ill patients. Once the mother’s condition is stabilized, the focus of attention is fetal delivery.

**Fetal Considerations**

In some instances, and of course most favorable for the fetus, AFE does not occur until after delivery. When AFE occurs before or during delivery, however, the fetus is in grave danger from the onset because of the maternal cardiopulmonary crisis. In addition to concern for fetal well-being, delivery of the fetus increases the chances for a good outcome for the mother because the weight of the gravid uterus on the inferior vena cava impedes blood return to the heart and decreases systemic blood pressure. Therefore, as soon as the mother’s condition is stabilized, delivery of the viable infant should be expedited. If resuscitation of the mother is futile, an emergency bedside cesarean delivery may be necessary to save the infant. Undeniably, the sooner after maternal cardiopulmonary arrest that the fetus is delivered, the more favorable is the fetal outcome. Therefore, as difficult as it may be, and even though the mother may be viewed as the primary patient, prolonged resuscitation efforts should be discouraged.

**Family Support**

Because the maternal and fetal mortality rates are so high, most likely an intensive care nurse will be called on to support the patient’s family members through crisis and/or loss. When the mother and infant are gravely ill, keeping their family members well informed and allowing as much access to the loved ones as possible are important. Warren reported that accessibility to physicians, unrestricted visits, and caring conveyance of adequate information were important to families’ satisfaction with the intensive care unit experience. When possible, providing and encouraging contact and interaction between parents and the newborn are essential in promoting parent-infant attachment.

In many cases, the mother dies but the infant survives. It is important to understand that an event that was anticipated with much hope and joy has gone terribly awry and that validating the wide range of emotions that the family might experience can

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**Table 5: Hemodynamic monitoring**

<table>
<thead>
<tr>
<th>Measures</th>
<th>Normal pressures</th>
<th>Abnormal pressure indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure (essentially the same</td>
<td>1-7 mm Hg</td>
<td>Elevated in right-sided heart failure, fluid overload, pulmonary hypertension, cardiac tamponade</td>
</tr>
<tr>
<td>as central venous pressure)</td>
<td></td>
<td>Decreased in hypovolemia</td>
</tr>
<tr>
<td>Pulmonary artery pressure</td>
<td>20-30/10-15 mm Hg</td>
<td>Increased pulmonary artery systolic pressure with right-sided heart failure, cardiac tamponade, pulmonary hypertension, pulmonary edema or embolus, adult respiratory distress syndrome</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure</td>
<td>6-15 mm Hg</td>
<td>Decreased pulmonary artery systolic pressure with hypovolemia</td>
</tr>
<tr>
<td>Cardiac output (heart rate x stroke volume)</td>
<td>4-8 L/min</td>
<td>Elevated with left ventricular failure, pulmonary hypertension</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>2.5-4.2 for adults</td>
<td>Decreased pulmonary artery diastolic pressure with hypovolemia</td>
</tr>
<tr>
<td>(cardiac output/body surface area, expressed</td>
<td>3.5-6.5 for pregnant women</td>
<td>Decreased with hypovolemia</td>
</tr>
<tr>
<td>in L/min per square meter)</td>
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</tbody>
</table>
facilitate grieving. In addition, the stages of normal grieving, anger and blame, can interfere with the normal parent-infant attachment and decrease the frequency of nurturing behaviors. Referral to counseling and support groups is most often helpful.

When both the mother and the infant die, it is important to allow the father and other family members time with their deceased loved ones. Staff members should be present to facilitate the initial contact and to answer questions, but they should also allow families to be alone. Pictures of the infant should be taken and offered to the family for keeping. Any infant clothing or blankets used, as well as infant identification bands, should be saved and given to the family. Many obstetric units have special “memory boxes” specifically for this purpose.

Summary

AFE is an unpredictable, unpreventable, and, for the most part, an untreatable obstetric emergency. Management of this condition includes prompt recognition of the signs and symptoms, aggressive resuscitation efforts, and supportive therapy. Any delays in diagnosis and treatment can result in increased maternal and/or fetal impairment or death. Whereas once the invariable outcome of AFE was death of the mother, today the prognosis is somewhat brighter thanks to increased awareness of the syndrome and advances in intensive care medicine. In any case, intensive care nurses are called on to provide physical, life-saving care to the patient and her fetus. Both during and after the event, supportive care must be administered to the patient’s family members, who are dealing with crisis and loss.

References
32. Dvorak-King C. PA catheter numbers made easy. RN. November 1997;60:45-49.
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